appearance, he completely lost the power of locomotion, and to side, and of moving his toes. The skin remained quite sensible to
continued natural, and he had neither headache nor giddiness".

It is difficult to avoid the diagnosis of acute polyradiculoneuropathy in this patient. He probably displayed at least seven of the
diagnostic criteria formulated by the National Institute of Neurological and Communicative Disorders and Stroke, and there
was the history of gastrointestinal illness. Wardrop could not have described loss of tendon jerks because their significance was not to
be realised until about 40 years later.1 Lumbar puncture for CSP
examination was not introduced for another 57 years.4 Landry’s
original description of “ascending paralysis” was in 1859. The first
report of the CSF findings by Guillin, Barré, and Strethol was
published in 1916, and this was amplified later.4

It would seem that Wardrop should have credit for the earliest
clinical description of this disease.

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TETRAHYDROAMINOACRIDINE AND THE CENTRAL ANTICHOLINERGIC SYNDROME

Sir,—I was intrigued by your editorial (Jan 17, p 139) on the
cholinergic treatment of Alzheimer’s disease via oral tetrahydro-
aminoacridine (THA). Before the availability of short-duration,
non-depolarising muscle relaxants THA was often used during
general anaesthesia to prolong the very brief action of
suxamethonium to 15–20 min. Nowadays parenteral THA is only
available on a named patient basis, and oral THA has never been
available in the UK. When I regularly used THA to prolong the
action of suxamethonium I formed the clinical impression that
confused and elderly patients could sometimes be strikingly
briskly mentally in the postoperative period for a day or so. It is
interesting to speculate, in the light of your editorial, whether this
increased mental alertness might have been a consequence of
restored levels of cholinergic activity in the brain, though there are
other possible explanations.

The antimuscarinic properties of hyoscine and atropine make
drugs valuable premedicants before the induction of general
anaesthesia. Atropine has been used clinically in the postoperative
period for many years. For this reason anaesthetists traditionally
eschew the use of hyoscine in patients over the age of 60 or 65.
Older patients are usually given atropine instead but perhaps a better choice is
glycopyrrolate, which has only peripheral anticholinergic effects in
premedication dosage. Apart from these considerations there is
generally little awareness amongst anaesthetists that they might be
helping patients by the unnecessary use of anticholinergic and possible
agonists of neuroeffector activity. The term “agonist” as applied to
the central nervous system (CAS) can be precipitated by anaesthetists has been
emphasised by Ruppreht and Dworacek.2 The mental impairment associated with
CAS ranges from simple memory disturbance to delirium and coma. The prescribed treatment is the careful intravenous
administration of physostigmine salicylate. I have found this
treatment effective in patients with arteriohyponxia who have remained
confused and confused 60–120 min postoperatively after apparently
adequate reversal of muscle relaxation and uneventful general
anaesthesia. The neostigmine used to reverse muscle relaxant drugs
does not cross the blood-brain barrier, unlike physostigmine, and
would not be expected to remedy the CAS. There is clearly an
important differential diagnosis to be made by the anaesthetist
between the confused agitation of incomplete reversal of the muscle
relaxant, the continued effects of other anaesthetic agents, and the
onset of CAS. Recognisable mental changes associated with CAS
can last a day or much longer in susceptible patients so that the
careful administration of physostigmine in these patients is justified.
It would be interesting to know whether THA, which can also cross
the blood-brain barrier, might be a suitable alternative parenteral
therapy. Peripheral muscarnic side-effects caused by both drugs
can be blocked with glycopyrrolate. The discomforts and possible
hazards of administering anticholinsterases parenterally make it
essential to consider very carefully whether a patient has CAS
before beginning treatment.

Further investigation of CAS is urgently required before it
decrees deeply engrained in the literature of anaesthesia. One
objection to the concept of CAS as a complication of anaesthesia is
that there is a multitude of neurotransmitters involved in the
ascending pathways that alert the cerebral cortex, all of which may
be altered by the complex effects of general anaesthesia.

CENTRAL ANTICHOLINERGIC SYNDROME

TETRAHYDROAMINOACRIDINE

The mental impairment associated with

ALZHEIMER’S DISEASE AND TETRAHYDROAMINOACRIDINE

EICOSAPENTAENOIC ACID IN FAT

SIDO,—The health effects of the m3-polyunsaturated fatty acid,
eicosapentaenoic acid (EPA; 20:5[n-3]), are of great interest.
Epidemiological investigations, however, are hampered by
problems in determining long-term dietary intake of EPA. Dietary
survey methods are imprecise and data on EPA in foods are scarce.
For the assessment of long-term individual intake of linoleic acid
(C18:2[n-6]), fat tissue microbiopsy has proved a reliable
alternative to survey methods, and a similar method for EPA would
be useful. Dr Wood and colleagues, in their paper on tissue fatty
acids and coronary heart disease (Jan 24, p 177), state that EPA is
not present in adipose tissue. We have now found that EPA can be
detected in fat tissue microbiopsy specimens by capillary
gas/liquid chromatography (figure). In three healthy volunteers (a
BODYBUILDER'S PSYCHOSIS

Sir,—Anabolic steroids are sometimes used by athletes seeking gains in muscle size and strength.1,2 Although the medical effects and risks of these drugs are well documented, their psychiatric effects are largely unexplored.3,5

We have treated two men who required hospital admission for psychiatric episodes apparently associated with use of anabolic steroids. A 40-year-old man had been prescribed methyltestosterone 10 mg twice daily for idiopathic impotence. Within 2 weeks he had symptoms meeting DSM-III criteria4 for major depression, together with delusions of reference and visual hallucinations. A 22-year-old construction worker and bodybuilder took two 8-week courses of methandrostenolone 15 mg daily. After the second course he had severe depressive symptoms. Although the depressive symptoms gradually lifted after several months, persistent paranoid and delusional ideas ensued.

Extensive medical and neuroendocrine workup of both patients was unremarkable. Both responded well to neuroleptics and were able to discontinue these medications permanently after several weeks. Neither patient reported any serious psychopathology before the index episode. Both, with no further exposure to steroids, have remained psychologically normal during more than two years' follow-up. Thus, anabolic steroids emerge as a likely causative factor in both cases.

Intrigued by these observations, we interviewed thirty-one other anabolic steroid users (twenty-nine men), recruited by

advertisements at gymnasium in the Boston and Los Angeles areas. Psychiatric diagnoses were made using a structured clinical interview.7 Three men had had psychotic symptoms with steroids: one reported auditory hallucinations of voices and noises lasting 5 weeks; another described delusions of reference and possible thought broadcasting; a third reported paranoid delusions. At least four others had "subthreshold" psychotic symptoms, including referential thinking, paranoid jealousy, and grandiose beliefs. For example, one man, while on steroids, believed that he could not be injured if he fell from a third-floor window. All seven experienced such symptoms only when taking orally active 17-alkylated steroids (methandrostenolone, oxandrolone, and/or oxymetholone), alone or in combination with parenteral preparations. None described any psychotic symptoms, even at the subthreshold level, at any time before steroid use.

Equally impressive was the observation that four interviewees met criteria8 for manic episodes while taking anabolic steroids, and five had major depression, either during or just after a course of the drugs. Manic-like behaviours were prominent; one 23-year-old twice bought expensive cars during courses of steroids and then was forced to sell them afterwards. Another subject, taking methandrostenolone, deliberately drove an old car into a tree at 60 km/h while a friend videotaped it. A third man, when "cut up" by another driver in Boston, pursued and cornered the offender and smashed his windshield with a crowbar. None of these subjects described comparable behaviours when not using steroids.

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REFERENCES


BACK PAIN AND TESTICULAR GERM CELL TUMOURS

Sir,—Dr Taylor (Feb 28, p 515) is correct in stating that germ cell tumours with no evidence of a testicular primary are rare, but patients are notoriously shy about reporting testicular abnormalities1 and even locally advanced tumours could escape notice during a routine orthopaedic examination. Oncologists have learned that back pain in cancer patients can indicate relapse in the retroperitoneal lymph nodes.2 Relief of pain is then a useful clinical marker of response to treatment.

We have found that severe, persistent back pain is the presenting feature of many malignancies in young people, including lymphomas, testicular tumours, and carcinoma of the cervix (to be published). Retroperitoneal lymphadenopathy can usually be demonstrated in these patients by simple ultrasound examination. It is important that the association between back pain and malignant lymphadenopathy is more widely recognised so that these potentially curable tumours are detected early.

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